ACROPACHY, OR SECONDARY SUBPERIOSTEAL NEW-BONE FORMATION.

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Our attention has recently been directed in a rather startling manner to the condition commonly known as hypertrophic pulmonary osteoarthropathy. A patient, whose history I shall give in some detail, returned to the clinic following an operation of subtotal thyroidectomy, having developed a most exaggerated form of clubbed fingers and swelling of the lower legs. Roentgenographic studies of the bones revealed a remarkable picture of subperiosteal new-bone formation, involving most of the long bones of the skeleton, but showing a change in the bones of the hands which had not been seen by the clinicians or the roentgenologists at the Johns Hopkins Hospital. After seeing in an article by Holthusen[1] the print of an X-ray of a hand showing similar changes, I have come across quite a number of articles, particularly in the German literature, which discuss this condition at great length. The cause of the change in the bones remains obscure, but our case throws some new light on the etiology and for that reason seems to me to be of unusual interest.

W. C. Age 22, colored, male. History No. U-9180. Admitted to the Johns Hopkins Hospital November 30, 1926.

For two years previous to his entrance to the hospital the patient had suffered from increasing nervousness. This was associated with a tremor which, after about nine months, caused him to abandon his work as a painter. At this time palpitation of the heart was very annoying and often kept him awake at night. He was restless. Although he was becoming noticeably thinner, his neck increased in size and he changed to a larger sized collar on three separate occasions. His eyes became poppy. In September, 1926, he came down with tonsillitis, which kept him in bed for three weeks and left him with very marked shortness of breath on exertion, swelling of the ankles in the evening (although they would be quite normal by morning), and a cough with yellow sputum. The cough, combined

with a ringing in the ears, prevented sleep for about three weeks before admission. In two years he thinks he has lost altogether 65 pounds in weight although his appetite has been good. He has had a moderate diarrhoea for the past two months. During the month of August he took one drop of iodine a day without apparent effect on his condition.

The Past History is uninteresting, except for frequent attacks of colds and a case of gonorrhea five years ago (1921). There is no history of primary or secondary syphilis.

Physical Examination: (By Medical Interne, J. H. H.) Temperature, 99.8; pulse, 124; respirations, 28; blood pressure, 156/78; height, 5 feet, 8 inches; weight, 110 pounds.

Patient is more than 40 pounds underweight; emaciated in appearance. Very restless, with a staring expression.

The skin is hot and moist; nail beds are pale.

Extremities: There is a fine tremor of the fingers. No clubbing of the fingers. No evidence of weakness. No tenderness over any bone or joint.

Deviation of third thoracic vertebra to the right. Ankles are swollen moderately with pitting edema which extends up over tibiae. The edema of the ankles is rather firm and pits with difficulty in spite of marked swelling.

Lymph glands: None palpable, except one in the right groin—1.5 by 1 cm.

Eyes: Marked exophthalmos; jerky movements, marked lid lag. Convergence normal. Tremor of closed eyelids; widening of palpebral fissure on focusing vision. Pupils react normally. Eye grounds normal.

Nasal septum intact.

Teeth in good condition.

Tonsils large; mucopurulent discharge on postpharyngeal wall.

Thyroid gland is markedly enlarged; more so on the right than left. Palpable thrill over right upper pole and to and fro murmur heard over gland. Supraclavicular fossa is filled by the thyroid gland.

Lungs: Clear, no râles.

Heart: second interspace, right 5.0, left 5.0; third interspace,

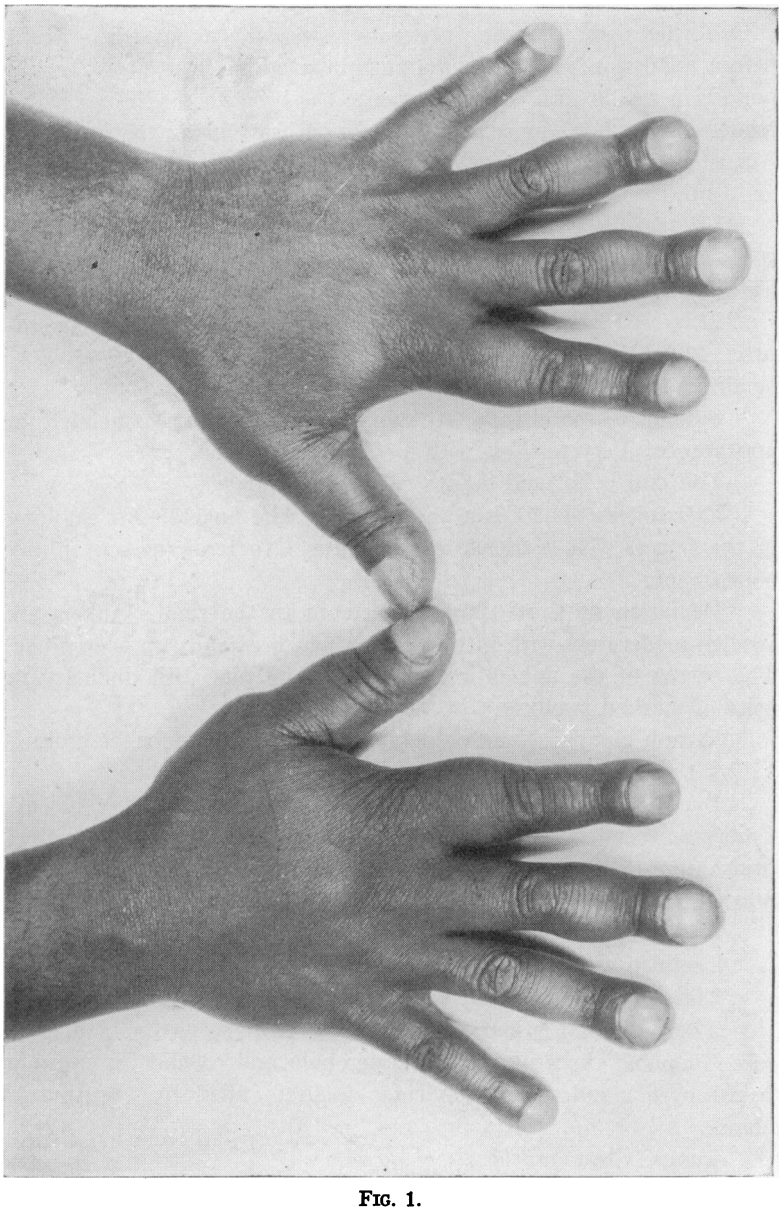

FIG. 1.
Clubbed fingers with swelling of the soft parts symmetrically distributed in the two hands.

right 6.5, left 7.0; fourth interspace, right 7.0, left 8.5; fifth interspace, left 10.0.

Pulse bounding, quick, regular in force and rhythm. Apex heaving. No thrill felt; no murmur heard.

Abdomen: No masses, no tenderness. Liver not felt; dullness extends 1 cm. below costal margin. Spleen not felt.

Genitalia: Normal.

Reflexes: Equal and active. No disturbances of sensation.

Laboratory Studies. Blood counts: R.B.C., 3,320,000; Hb., 60%; W.B.C., 7,610.

Differential: P.M.N., 73%; P.M.E., 1%; S.M., 11%; L.M. & Tr., 14%; unclassified, 1%.

Slight anisocytosis; platelets look normal. Stool negative.

Urine: Sp. gr. 1018; reaction acid; albumin +; sugar 0. Microscopic: Few w.b.c.

12/1/26. *X-ray Examinations.* Teleoroentgenogram: M.R. 6, M.L. 8.5.

Chest: No evidence of substernal thyroid. Lungs clear, except for non-tuberculous infiltration at right base. Heart a trifle enlarged, particularly second curve to left.[2]

12/2/26, *Basal Metabolic Rate* +59 (poor test). Temperature 101.8 (rectal); weight 110 pounds.

12/2/26. *Gynergen Test:* (ergotomin). No subjective discomfort.

12/2/26. *Electrocardiogram:* Rate 110, P-R interval .16 second. Normal mechanism.

12/2/26. *Pulv. Digitalis* 0.9 gram today; 0.2 gram q.d. for 14 days.

12/5/26. *Lugols Solution* minims xxx every day for 12 days.

12/11/26. *Blood count:* R.B.C. 4,450; Hb. 78%.

12/15/26. *Basal Metabolic Rate* +41% (poor test). Temperature 99 (rectal); weight 127 pounds.

12/17/26. *Pulv. Digitalis and Lugols Solution* discontinued.

Ergotomin 0.5 mg. twice daily for two days, without effect.

12/19/26. Lugols Solution minims xxx every day for nine days, without change in pulse rate (120).

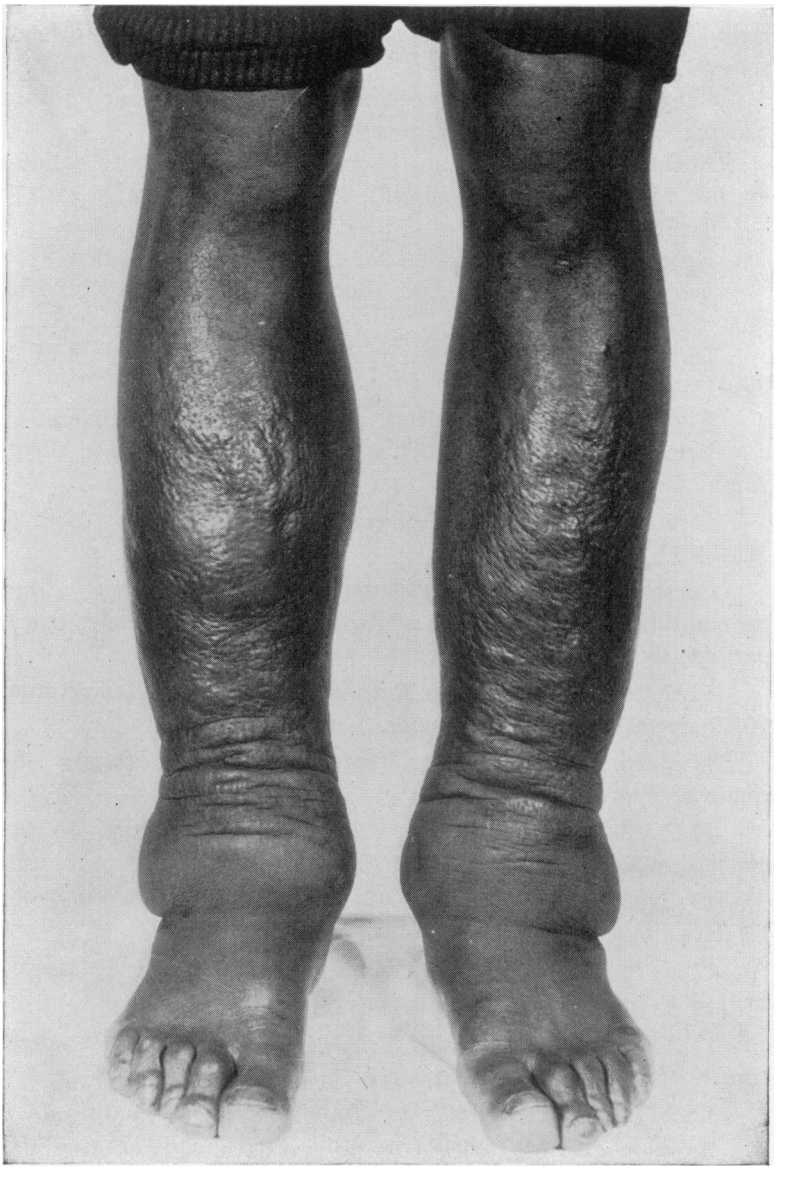

FIG. 2.
Brawny edema of the legs.

12/28/26. *Operation.* Subtotal thyroidectomy. Gland was hard, slightly irregular, but rather vascular.

Pathological Report: Exophthalmic goiter.

Severe post-operative reaction with temperature 105 degrees, pulse 184 for two days, after which temperature and pulse gradually fell in five days to normal for the first time during patient's stay in the hospital.

Basal Metabolic Rate —3%. Weight 136 pounds.

1/16/27. *Discharged.* Improved.

Following his discharge from the hospital the patient moved to Chicago and was not seen again until 2/15/29.

Interval History: After leaving here in 1927 the patient lived several weeks in Baltimore, a few months in New York City and then went to Chicago, but has not worked at all. Eight months after leaving the hospital he began to notice that whereas his ankles had theretofore been a little swollen at night, they were staying swollen constantly and enlarging. About the same time he noticed the ends of his fingers were enlarging and becoming curved, although not painful. The swelling of his fingers and ankles progressed slowly until at the present time it involves the wrists and extends from ankles to knees, leaving the feet unaffected. The only treatment has been rubbing with alcohol. The patient complains of no other symptoms at the present time, except that his eyes bother him occasionally when he reads. There has been no pain, except upon vigorous movement of the hands and legs which has, however, been the cause of his not working. He gained about 30 pounds in six months after leaving the hospital, but has lost weight since then.

Physical Examination: (H. M. T., Jr.).

Well nourished.

Skin dry. Palms slightly moist; axillae quite moist. Hair is normal in texture, slight thinning over crown.

Eyes: Very marked bilateral exophthalmos. Upper lids slightly swollen. Convergence poor.

Ears: Watch tick heard normally.

Mouth: Tongue broad and flat; moist. Teeth in good condition. Tonsils not enlarged.

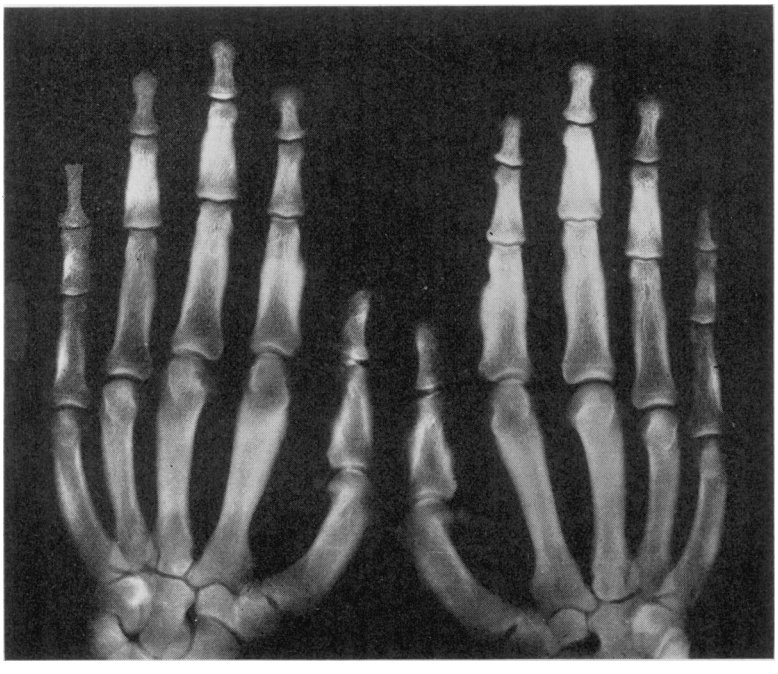

FIG. 3.

Roentgenogram showing tufting of the terminal phalanges and marked subperiosteal new-bone formation symmetrically distributed in the two hands and underlying the soft tissue swelling, seen in photograph taken two years after subtotal thyroidectomy.

Trachea in midline; thyroid not palpable, well-healed thyroidectomy scar.

Glands: No general glandular enlargement.

Lungs clear to percussion and auscultation.

Pulses equal and synchronous. Regular in force and rhythm. Rate 60 per minute.

Heart: Sounds clear at apex and base. The second aortic is loud and plupping. Blood pressure 150/110.

Abdomen: Liver and spleen not felt. No masses.

Genitalia: Phallus and gonads are normal. Slight left varicocele. Inguinal glands slightly larger than normal.

Reflexes: Very active. Chvostek and Trousseau signs are negative.

Extremities: There is an extreme degree of clubbed fingers with enlargement of the hands and wrists. There is a thickening and bowing of the left second metacarpal and also a thickening of the soft parts around all of the middle phalanges. There is thickening of the wrists. Elbows and shoulders are normal. The feet are approximately normal. There is excessive edema from top of low shoes to the knees. The skin of this area is brawny and pigmented.

Impression: The patient probably has slight post-operative myxedema. The interesting feature is the hypertrophic osteoarthropathy, a possible explanation of which may be found in the alteration of the circulation following a sudden reduction of the metabolic rate.

Laboratory Studies. Urine: Clear, medium amber, 1027, acid; sugar 0, albumin trace, few hyalin casts, few epithelial cells and w.b.c.; no r.b.c.

Blood counts: Hb., 91%; R.B.C., 4,720,000; W.B.C., 14,650.

Differential: P.M.N., 73%; P.M.E., 4%; P.M.B., 1%; S.L., 20%; L.L., 2%.

X-ray Report: Lungs clear. Heart and aorta normal in size. M.R. 5, M.L. 9.5.

Metacarpal bones very short; few periosteal changes in bones of hands and legs, otherwise normal.

2/15/29. *Basal Metabolic Rate* —9% (perfect test). Weight 177 pounds; pulse 56.

2/15/29. *Electrocardiogram:* Rate 52; rhythm sino-auricular; P-R interval .19 second. T waves all upright. Normal sinus rhythm. Sinus bradycardia.

2/18/29. *Blood Chemistry:* Nonprotein nitrogen, 32 mgm. per 100 c.c.; calcium, 11.4 mgm. per 100 c.c.; phosphorus, 4.0 mgm. per 100 c.c.

The patient was requested to enter the hospital for observation and treatment, but declined and disappeared from sight until:

4/9/31. *Interval History:* Since last seen patient has worked spasmodically as a tailor's helper, but has not felt well. He has noticed no change in the size or shape of his hands and legs. Last January he fell, injuring his back and right leg and he is still shaky and nervous from the fall, and has pain in the left flank.

4/9/31. *X-ray Report:* Marked new-bone formation of the bones of the hands and feet. Unable to diagnose.

4/14/31. *X-ray Report:* Marked periosteal changes in the bones of the hands. Long bones, head and spine normal.

Wassermann reaction: Negative.

Blood pressure 160/120 to 185/130. Same in two arms.

Laboratory Studies. Urine: Specific gravity, morning 1016, evening 1026; albumin, morning +, evening +; sugar, morning 0, evening 0; micro: a few w.b.c.; one hyalin cast.

Phthalein Test: First hour, 43%; second hour, 12%; total, 55%. Nonprotein nitrogen, 38 mgm. per 100 c.c.; total proteins, 7.95 mgm. per 100 c.c.; A/G ratio, 63/37; calcium, 10.2 mgm. per 100 c.c.; phosphorus, 3.7 mgm. per 100 c.c.; cholesterol, 230 mgm. per 100 c.c.

4/24/31. *Basal Metabolic Rate* —20% (perfect test). Pulse 54; weight 187 pounds.

4/24/31. *Thyroid Medication* begun, grains I a day. After 18 days patient reported feeling much better. No pain in the back and pulse 62. Thyroid extract grains II after 14 days produced "quivers around the heart."

The capillaries of the nail bed were examined microscopically by Dr. J. Evans who found an unusual condition. There was great dilatation of the venous side of the capillaries and the arterial side showed an extreme degree of tortuosity. The capillary pressure was slightly elevated.*

Thyroid extract grains 1½ a day for a month produced very little change and the patient was annoyed by insomnia, ache around his eyes and great fatigue in the afternoon, requiring two hours sleep.

9/22/31. Patient took thyroid steadily up to two weeks ago. Has felt some better with no noticeable change in his hands and legs.

Summary of Case.

A young colored man of 22 years was admitted to the hospital in November, 1926, suffering from a rather severe form of diffuse goiter with hyperthyroidism which, judging from the history, had existed for two years. Following the usual treatment and operation,

*Holger[3] says in his article, "Interesting but inconclusive is the finding of normal capillaries in clubbed fingers *in vivo* in one case."

the basal metabolic rate fell from the admission level of 59% above normal to 3% below normal and he gained 26 pounds. After his discharge from the hospital he gained about 30 pounds more in eight months. At the end of this time he noticed clubbing of the fingers and a change in the swelling of his ankles which, previously transitory, had become constant and firmer. These conditions increased gradually, but with only slight pain and that only on vigorous movement of the hands. In 1929 the basal metabolic rate was —9% and the X-ray showed laying down of new bone under the periosteum of the bones of the hands and feet and long bones of the extremities. In April, 1931, the basal metabolic rate was —20% and the new-bone formation had become more extensive. A diagnosis of post-operative hypothyroidism with secondary hypertrophic osteoarthropathy was made and thyroid replacement therapy instituted with definite improvement in the subjective symptoms. No convincing change has been noted in the bones since the patient began to take thyroid, although an apparent thinning of the sub-periosteal new bone has been seen at the end of three and a half months.

Discussion.

This is the first reported case of clubbed fingers with sub-periosteal new-bone formation occurring in association with disease of the thyroid gland. The changes in the bones of the hands are as extensive as any described in the literature and the swelling of the legs is greater. The new bone is confined to portions of the skeleton which are covered by periosteum and is not seen in the joints. It is greatest in the midportion of the short bones, but involves the entire length of most of the long bones as well. There is also well-marked increase in the tufting of the terminal phalanges of the fingers. Because the swelling of the soft parts in this case occurs so clearly above the bony changes, even including the tufting of the terminal phalanges, it has occurred to us that the clubbed fingers which are seen in this condition may represent a different soft tissue change from that of clubbed fingers which occur without any underlying bony change.

Most of the cases acquire the new bone in thin layers under the periosteum, several layers becoming visible in the X-ray as time goes on. Our case, however, shows a piling up of new bone with a

fuzzy surface to a thickness of two or even three millimeters. This difference, I think, is due to the rapidity of the process. The swelling of the soft parts in the hand (and presumably elsewhere as well) occurs over the bony swelling as though it were part of the same process. One extraordinary feature of the condition which has been commented on before (Fraenkel)[4] is the symmetry of the process. The same portion of the same bones in the two hands is involved to approximately the same degree. Hitherto this condition has been described as occurring secondary to suppurative intrathoracic lesions, mediastinal new growths, tumors of the lung, liver abscess, pyelonephritis, cirrhosis of the liver with jaundice, certain obstructive lesions of the gastrointestinal tract, possibly syphilis, and perhaps incorrectly, to congenital heart disease. Our case presents none of these features.

In 1889 Bamberger[5] reported two cases of bronchiectasis which developed clubbing of the fingers and toes and gross changes in the bones of the lower legs. He attempted to reproduce the bony changes in rabbits by injecting the patient's sputum into the animals on the theory that the etiological factor was a toxin liberated by the suppurative process.

The following year Pierre Marie[6] described a syndrome which he called hypertrophic pulmonary osteoarthropathy (osteoarthropathie hyperthrophiante pneumique) consisting of widespread enlargement of the bones and clubbing of the fingers in cases of heart and lung disease. He was careful to differentiate this condition from acromegaly, although his best case, seen in the light of our further knowledge, undoubtedly combined certain features of acromegaly with hypertrophic pulmonary osteoarthropathy. Later on much interest was aroused by the constant accompaniment of clubbed fingers (Trommelschlägelfingern) with the bony changes, as this secondary enlargement of the distal phalanges of the fingers and toes had been noted by Hippocrates. Trousseau,[7] after several centuries during which it had been overlooked, described clubbed fingers accurately and pointed out their association with advanced pulmonary tuberculosis, empyema, emphysema, nervous asthma and organic heart disease.

After the appearance of Bamberger's article in Germany and Marie's in France, clinicians in all countries reported cases secondary

to various diseases, among which were tuberculosis, bronchiectasis, empyema, lung abscess, lung tumor, lymphoblastoma, organic heart disease, syphilis, cirrhosis of the liver with jaundice and amebic hepatitis, with a few apparently spontaneous cases. Schmidt[8] in 1899 collected the literature on the subject. He concluded that clubbed fingers with the change confined to the soft tissues represent an early stage of what may or may not further develop into the more advanced and rarer form with bony thickening. He thought that the joints are never involved and that the greatest swelling occurs in the middle portion of the shaft of the long bones. Most of the bones of the skeleton may show changes, including the clavicle and ilium.

In this country, Thayer,[9] Janeway,[10] Landis[11] and others have written on the subject and in 1915, Locke[12] summarized the literature and added five carefully studied cases. He considered the clubbed fingers and bony swelling as part of the same condition and showed that early subperiosteal changes could be demonstrated in all of his cases of clubbed fingers.

Fraenkel[4] in 1917 reported seven additional cases and argued against the earlier impression (agreeing on this point with Schmidt) that the joints and peri-articular tissues (cartilage) are commonly involved. None of his cases studied at autopsy showed any such change and he conceived the condition to be a form of hyperplastic periostitis which involves characteristically the mid-bones of the hands and feet and the mid-portion of the long bones. The underlying old bone may be unchanged or may show thickening or thinning.

A splendid article on the subject was published in 1920 by Högler[5] from Falta's clinic in Vienna. He reported five cases, one of which was merely a case of clubbing of the fingers, and he carefully analyzed the case reports from the previous literature. If his conclusions are correct, and I must say that in most respects his points are very well taken, we must readjust some of our ideas on this subject.

In an effort to eliminate the obvious disadvantages of most of the names which have been applied to this condition, such as chronic hypertrophic pulmonary osteoarthropathy, hyperplastic periostitis, ossifying periostitis, secondary ossifying periostitis, toxic periostitis, toxicogenic osteoperiostitis occificans, etc., he suggests the name acropachy. By this name he avoids pathological descriptive terms

which are likely to be incorrect (there is no evidence of inflammation of the periosteum, and the joints are rarely involved), he avoids terms suggesting the etiology which are also likely to be incorrect or inconclusive and hints at a similarity to acromegaly with which the condition was first confused.

The bone change consists of a deposition of new bone between the cortex and periosteum of the long bones, particularly the lower ends of the bones of the forearms and lower legs and metacarpal and metatarsal bones. In extreme cases similar changes have been described in the clavicles, ribs, pelvic bones, scapulae and malar bones, and even in the transverse processes of the vertebrae. The joints are never involved in the original process, although involvement of the end of the bone may lead to hindrance of the joint. The underlying bone shows no change except that seen following disuse. Hystologically the cortical part of the bone is very little, if any, changed (Schlagenhaufer).[13] Högler disagrees with Sternberg who described evidence of inflammation in the periosteum itself. More and more, he says, it looks like a hyperplastic process of the periosteum. This has a true analogue in the osteophyte building during pregnancy, but is not a true inflammation.

Often there is swelling of the wrists and ankles and the latter swelling may even amount to a brawny edema.

The condition may be painful or accompanied by very tender points over the affected areas.

Although usually occurring in cases with clubbed fingers, several such cases have been described without clubbed fingers and also innumerable cases of clubbed fingers have occurred without any bony changes. For these reasons it appears that this syndrome is quite distinct from that of clubbed fingers, although the two conditions probably are caused by the same variety of underlying morbid process.

On careful analysis, most of the authentic cases were found to occur in patients suffering from some collection of pus in the chest or some form of mediastinal new growth, usually lymphogranuloma. Högler doubts whether any true case has occurred secondary to congenital heart disease without accompanying pulmonary disease. Unilateral involvement of one arm has been described in cases of aneurysm of the subclavian artery and in trauma to the brachial

plexis. A number of characteristic cases have been observed following biliary cirrhosis of the liver with jaundice. Finally a small number of cases with no obvious etiology may be found in the literature.

Högler thinks it very interesting that most cases follow a purulent process in the thoracic cavity or a mediastinal growth from which some toxic substance may emanate. One of the cases he reported of lymphogranuloma with a mass in the mediastinum was treated by irradiation of the mediastinal mass. Along with the general clinical improvement the bony changes, which had been extreme, faded away until hardly noticeable in the X-ray films. Högler points out that Gerhardt,[14] Libermann,[15] Ruhle[16] and V. Hoffmann[17] wished to give chief etiological importance to congestion, but he says later on that congestion plays no important rôle since we no longer include the cases of heart failure as a special group and since there is another group of cases with high grade acropachy in whom stasis in the large and small circulation is entirely precluded.* After considering the possible influence of toxins on trophic nerves in the periosteum, the possibility of an irritating substance which perhaps can be generated by malignant tumors, etc., Högler finally concludes that we must clearly admit that this hypothesis really is nothing but another way of writing down the important facts which depend upon various observations and experimental data.

Schirmer[18] in 1923 published an autopsy report on one of Högler's cases which subsequently died of "lymphoblastoma" or Hodgkins disease. There existed infantilism with a normally formed but extremely underdeveloped thyroid gland and with a normal hypophysis. He refers to Franchini[19] who in 1911 reported a case of putrid bronchitis with atrophy of all the endocrine glands and infantile habitus. Braun[20] found an adenoma or some other lesion of the hypophysis in three of four studied cases of this condition and suggested that the bony changes are caused by a dysfunction of the pituitary gland.

Our case presents several very interesting features. In the first place it developed shortly after the patient had been converted quite

*I should like to suggest that a slowing of the entire blood flow may occur without local evidence of congestion and yet still have its effect on such tissues as periosteum, even if the generally accepted connection between congenital heart disease and acropachy is incorrect.

suddenly from a condition of hyperthyroidism to one of hypothyroidism by iodin medication followed by subtotal thyroidectomy. Careful search before operation and on many occasions since operation, over a period of five years, has failed to reveal any other disease process either in the thoracic cavity or elsewhere in the body. We are forced then to conclude that in this case the changes were in some way produced by rapid alteration in the patient's endocrine balance. No such changes have been observed in exophthalmic goiter or in myxedema. Long standing cases of hyperthyroidism do exhibit generalized osteoporosis and conversely myxedema tends to produce a widespread increased density of the bone, but without change in the size of the bone and without new-bone formation. The changes of the bones in parathyroid disease are very striking, but, there again, they differ markedly from our case by involving the original bony structure in a condition known as osteitis fibrosa cystica. No indication of parathyroid dysfunction in our case could be brought to light and the blood calcium and phosphorus determinations were always normal. That one or more of the other glands of internal secretion were altered functionally by the operation on the thyroid gland seems likely, but I am unable to connect such changes with the development of new bone seen in our case. The thymus gland is frequently found enlarged in cases of exophthalmic goiter. That the thymus should assume such proportions as to be considered a mediastinal tumor, which might produce pressure and yet not be visible in the X-ray of the chest seems doubtful but barely possible. Pressure symptoms in an adult from an enlarged thymus are rarely, if ever seen, although the thymus is usually enlarged in hyperthyroidism. No involvement of the pituitary gland was demonstrable by X-ray examination of the sella turcica, by determination of the visual fields or by sugar tolerance tests. We infer that a change in the functions of the pituitary gland probably accompanies changes in the function of the thyroid gland, but we are dealing in our case with a skeletal deformity quite different from acromegaly or gigantism.

In the radiographs of the pelvic bones certain areas of rarefaction faintly suggested Paget's disease, but examination of the other bones, particularly the skull and spine, did not bear that out and

changes like those seen in the bones of the hands in our case have not been described in Paget's disease.

We might quite properly, it seems to me, place some importance on the sudden change in blood flow which we know to have taken place in this case when the hyperthyroid condition was abruptly converted into one of hypothyroidism.[21] In a young adult this sudden slowing of the circulation has for some reason been accompanied by the remarkable abnormality described above. The perfect bilateral symmetry of the lesion requires an explanation. What condition, we must ask ourselves, might affect the bone or periosteum of the two arms in such an irregular but identical manner? Would not a circulating toxin call forth a similar response in all of the proximal phalangeal bones instead of selecting the mesial surface of each second proximal phalangeal bone to concentrate its attentions on? Alteration of the circulation, on the other hand, might easily affect one finger, let us say the index finger, before the others, and this, of course, would be true for both sides alike. In our case, as in those of Högler, a persistent, edema-like swelling of the lower extremities is strong evidence in favor of local congestion. One is led to wonder whether the common factor in all of these cases may not be some change in the circulation. Such a change occurs in cases of jaundice with bradycardia and might also occur if a fraction of the lung substance ceased to act properly in its function of aerating the blood. Mechanical pressure from a mediastinal mass does interfere with the blood flow in the great vessels and the disappearance of the bone changes in such a case following deep X-ray therapy to the mediastinum may be accounted for by the removal of such pressure. It would seem that the circulatory change must be abrupt and that its effect is most striking when it occurs in patients whose osseous system is still in the process of adolescence growth.

It is important to point out the obvious objections to considering circulating toxins the cause of acropachy. Why, for instance, should lung abscess produce such a toxin and abscesses elsewhere in the body not produce it? Why should it occur in its most advanced form with Hodgkin's disease which involves the glands of the mediastinum, and not occur with Hodgkin's disease confined to glands elsewhere in the body? Why should it occur with carcinoma of the bronchus and not with carcinoma in other parts of the body? Why

should this toxin be liberated by pulmonary tuberculosis and not by tuberculosis of other organs?

As may be inferred, the writer is not in favor of the circulating toxin theory. In all honesty, however, he must confess self-evident objections to the altered blood flow theory, also. Why do we not see acropachy in the many cases of unilateral femoral thrombophlebitis? As far as I know, subperiosteal new-bone may occur in these cases without ever having been noticed. If insufficient oxygen supply is an important feature, why has acropachy not been found in long standing cases of severe anemia? It may be, I think, that the tissue adjustment to an anemia is gradual, whereas in acropachy time for adjustment is lacking.

SUMMARY.

I have presented the case of a young colored man who developed diffuse goiter with hyperthyroidism for which subtotal thyroidectomy was performed. Eight months later he noticed clubbing of the fingers and brawny swelling of the legs and when examined two years later was found to have an extreme degree of acropachy. A careful examination made at the time of operation and repeated on two further occasions, four, and again five years after operation, failed to reveal other disease or lesion of heart or lungs. I found no mention of similar cases in the literature, but I have discussed the important articles on acropachy as it occurs in various other conditions. Nothing is known as to the mechanism of the bony change in this syndrome.

The new and unusual case I have reported adds strong evidence of the importance of altered blood flow in the production of subperiosteal new-bone formation. The only conclusion I am able to reach, after contemplating the many factors involved in cases of acropachy, is that altered blood flow offers itself as the most plausible common etiological factor.

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DISCUSSION.

DR. EDWIN A. LOCKE: The topic discussed by Dr. Thomas is extremely interesting and, as he has suggested, it is atypical in almost every direction.

There are one or two thoughts that occur to one, however. Perhaps I might ask Dr. Thomas whether anything was noted in this man as to whether his body

had shortened, as to whether there would be any suggestion that he might have had some atypical from of Paget's disease. I suppose the X-ray of the skull would certainly clear that question up.

One other question that Dr. Thomas raised was the possible appearance of such conditions as those being due to a congenital cardiac disease pure and simple. Campbell Howard had, last year, such a case as that. A man with a very profound congenital cardiac disease, who had extreme degrees of thickening of the fingers and toes, and in whom on autopsy nothing else was found except this congenital cardiac disease, no pulmonary lesion.

DR. JAMES ALEXANDER MILLER: This is a very fascinating subject. I am sure it has engaged all of our interest, and I think this presentation of Dr. Thomas is extremely valuable.

There are just two points that I would like to suggest: that is the fact that it is well know that there is such a thing as congenital club fingers and that it runs in families. Just what that is I think is a very interesting angle of the subject. Secondly, in connection with the slowing of the blood flow, last winter (I have now forgotten his name) a very prominent Englishman gave a paper in the Academy of Medicine in connection with changes in peripheral circulation, in which he brought out the fact that the capillary circulation in its terminal phalanges is very different from elsewhere in the body, that there is a direct immediate transition between the arterial and venous systems in wide channels, not the ordinary capillary flow at all.

It has seemed to me possible that these changes, so often in the soft tissues first, then bony changes later, might occur from the fact that that particular location had something to do with it and would be in line with Dr. Thomas' innocent hypothesis about the blood flow changes.

DR. HOWARD B. SPRAGUE: I would like to go on record as declaring that we do see cases of congenital club fingers without congenital heart disease, and then again we see cases of congenital pulmonary tuberculosis in which club fingers are typical.

We have seen a good many cases of the talipomanus and have studied some of these very carefully, and in all cases of this type with cyanosis I think club fingers are prominent. However, it is not really entirely clear whether one can blame the pulmonary circulation in this case on the theory of some people, when there is, in addition, in congenital heart disease of this type, peripheral capillary change.

One other thing is that in these cases of marked cardiac cyanosis they frequently do not have clubbed fingers, so that possibly this materially adds to the confusion as far as etiology is concerned.

DR. WILSON: The blood stream is very interesting but hardly explains all of our cases. I have one now under observation and have had for a number of years, who, twenty years ago, had a pulmonary tuberculosis lesion from which she made a complete recovery, but before recovering from her pulmonary lesion she developed an osseous lesion which persisted for several years before entirely

healing. In the course of these she developed these very pronounced club fingers, which she has retained from that time on.

It might seem that this possibly was a case depending upon a circulating toxin. That was my assumption at the time. It would be very difficult to say that this was dependent entirely upon an alteration of the blood stream and certainly not upon a sudden alteration.

DR. BRAY: What I have to say is really not highly important, but it must be twenty years ago that we studied this subject at Raybrook rather exhaustively, and we included at least 2000 cases or more, and I agree with the doctor who just spoke about congenital heart disease. I have seen a number of cases of this type when I was a student in Edinburgh under Dr. Gibson, and the few autopsies that I saw at that time rather confirmed the view that uncomplicated congenital heart disease was responsible for this condition, but in tuberculosis we have this very definite set of clinical manifestations. If we select those patients with pulmonary tuberculosis who have a long drawn-out clinical course, with more or less urgent dyspnea and marked cyanosis, presumably in these febrile cases we will find that a small minority will develop this condition, but a very large group, I should say at least 80 per cent, do not, and with these conflicting findings I think the question must arise that in the individuals who present these bony disturbances they must have some sort of a constitutional diathesis.

With this view in mind I took the question up with Professor Stockard at Cornell, who probably in this field is one of our leading authorities, and he thought that up to the present time we must be satisfied with that explanation, that some patients had this constiutional diathesis, which probably explains the development of pulmonary tuberculosis.

PRESIDENT HAMMAN: I should like to emphasize the resemblance in the X-ray of these lesions to those of syphilis, not so much of the fingers, but of the long bones. I think almost any roentgenologist would call the lesion syphilis.

I was asked to see a case in which the patient had changes of a lesser degree, but there were quite marked changes in the long bone. I was asked to see the patient simply because the roentgenologists insisted that it was syphilis and could be nothing but syphilis, and the internist knew better and knew it was not syphilis.

Would you care to close the discussion, Dr. Thomas?

DR. THOMAS: These points that have been brought up give me a little more time. It is a tremendously complicated subject, of course. I don't mean to say that I think that this case that we had, at all proves anything. However, here is a case in which the bony change was so extreme, more extreme in the hand than any case we have ever had at Hopkins, and in whom there was no possible portal of entry for any of the well-known toxic products, no alcoholism, which has also been one of the things that has been brought up, also no arsenic. Here was a case who did present a sudden slowing of the blood flow.

We do know, as Dr. Miller and Dr. Wilson have said, that certain cases

present club fingers in heart disease. If club fingers is the same condition, and it undoubtedly is caused by the same type of thing, then we are dealing with a circulatory manifestation pure and simple. That is all right, we can go that far and say the circulatory change can do it.

Our case had an endocrine disturbance we could also possibly say, but that is not the obvious answer. Perhaps tuberculosis is not the obvious answer. It is the sudden slowing of the entire blood flow. As Dr. Miller has pointed out, the capillaries in the terminal phalanges are different from anywhere in the body and produce clubbed fingers. The circulation in the hands is still different from anywhere and produces periosteum, possibly because the circulation toward the periosteum is less able to balance itself on a new level, and in a sudden diminution of the blood this new bone formation takes place.

Dr. Gordon Wilson asked about the height, and we have those figures because we did basal metabolic rates, and I don't know what they are, but I will look that up as soon as I go home.

We thought of Paget's disease, and the X-ray of the skull is perfectly normal.

DR. MILLER: How about congenital club fingers?

DR. THOMAS: I don't know. They are certainly something to think about. If that comes into the same group of circulatory changes we must consider that. The capillaries in this patient were studied under the microscope, and there were changes noted. The venous was very much engorged and swollen, and the arterial side was very tortuous. The arterial pressure was a little elevated in this case, and in cardiac cases with club fingers the pressure is normal or is diminished—at least I was told this by Evans, the man who studied this case. That that circulatory change might occur in the fetus or in the congenital lesions, which we know do produce club fingers, I was never in doubt about.